

**A red orange extract modulates vascular response after a recreational Dive : a pilot study on the effects of anthocyanins**

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*Publication date:*  
2015

[Link to publication](#)

*Citation for published version (APA):*

Balestra, C., Cimino, C., Theunissen, S., Snoeck, T., Provyn, S., & Virgili, F. (2015). *A red orange extract modulates vascular response after a recreational Dive : a pilot study on the effects of anthocyanins*. Poster session presented at The annual meeting of the European Underwater and Baromedical Society (EUBS 2015), Amsterdam, Netherlands.

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## Introduction

# A red orange extract modulates vascular response after a recreational Dive : a pilot study on the effects of anthocyanins

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Scuba diving, breathing either compressed air or specific gas mixtures, at recreational or professional level, has been reported to affect endothelial function, and to impair flow mediated dilation (FMD) and heart function. These effects have been frequently attributed to an “alleged” increased generation of reactive oxygen and nitrogen species. Accordingly, nutritional antioxidants have been proposed as an expedient strategy to reduce endothelial adverse effects of diving.

## Methods

16 volunteer subjects (12 males and 4 females,  $40,1 \pm 5,8$  yrs) certified experienced scuba divers, were enrolled in the study and randomly assigned to 2 groups. One group was administered with two doses of 200 mg of an anthocyanins (AC) rich extract from red oranges (Red Orange Complex-ROC<sup>TM</sup>), consumed 12 hrs and 4 hrs before the dive, respectively (the pills were swallowed drinking one glass of water); the other group received a placebo with the same amount of water. All subjects reported to a specifically designed indoor pool (Nemo 33, Brussels, Belgium) and performed a single “standard dive” at 34 m depth (4 Atmospheres Absolute-ATA) for 20 minutes observing the US Navy diving procedures. Blood samples were collected, FMD, and multi-frequency body impedance assessed just before diving and just after surfacing.

## Integrative



## Physiology

## Results

Overall, dive was associated with modest changes in hematocrit (HTC) and body hydration. However, at the end of the dive, a small but significant ( $P < 0.05$ ) difference of HTC was observed between non supplemented divers and subjects administered with ROC (see Figure 1). This was surprising, since it is known that divers presenting to hyperbaric recompression centers for DCS treatment are usually hemoconcentrated. A new model has to be proposed to understand such a discrepancy. We can hypothesize that during the dive a fluid shift occurs to compensate the immersion diuresis. This water would be like a “loan” to the vascular compartment. The following sequence is hypothesized: a progressive vascular compartment concentration, by immersion-induced diuresis is compensated - within a limited time window - by water shifting towards the vascular compartment from the extracellular compartment. This is advantageous during the off-gassing period since at that time the nitrogen gradient goes from the tissues to the vascular bed and the vascular dilution is useful to accept the increased nitrogen supersaturation. This can explain why divers without DCS are not always hemoconcentrated a short time after the dive. However, this is a temporary situation and data are available to show that a reversal of this fluid shift occurs at some later time points. Data collected let us speculate about the presence of two mechanisms induced by AC and affecting endothelial functions and water movements : one due to fast signals induced by the second ROC administration and possibly mediated by extracellular regulated kinase 1 and 2 (ERK1/2), and a second slower one (induced by the first ROC administration), due to the expression of genes involved in cellular response to high oxygen, pressure, nitrogen.... both affecting nitric oxide bioavailability and vascular function including internal fluid shifting.

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Figure 1: HTC changes (% of pre-dive value) after diving and effect of the administration of 400 mg ROC. \*  $p < 0.05$  according to Mann-Whitney U test vs unsupplemented

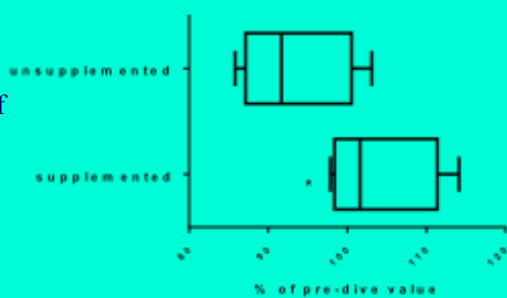


Figure 3: FMD changes (as % of the initial value) after diving and effect of the administration of 400 mg ROC. \*  $p < 0.05$  according to Mann-Whitney U test vs unsupplemented

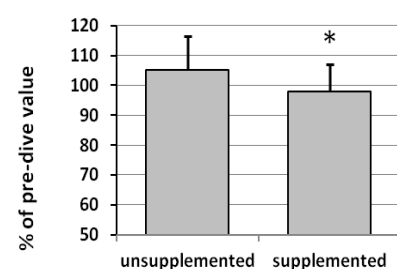
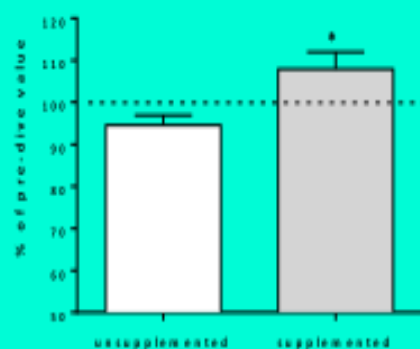


Figure 2: Extracellular water measured by multifrequency bioimpedance changes (as % of pre-dive value) after diving and effect of the administration of 400 mg ROC. \*  $p < 0.05$  according to Mann-Whitney U test vs unsupplemented

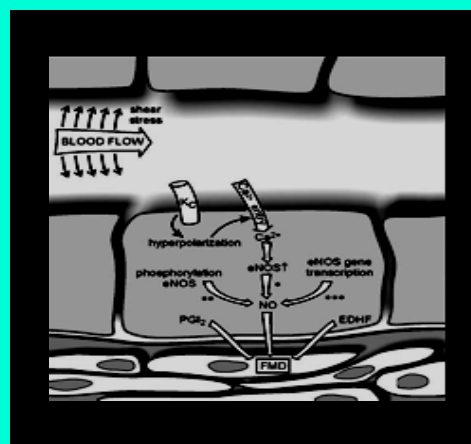


Figure 4: Possible scavenging effect on Reactive Oxygen Species (and probably Nitrogen Reactive Species) of anthocyanins preventing endothelial function impairment. (From: Moens et al., Chest 2005, 127: 2254-2263)

## Conclusions

Our data indicate that AC administration significantly prevents the potentially harmful endothelial effects of a recreational single dive. The mechanism underlying this protective activity can only in part be explained by the antioxidant capacity of AC and possibly involves the modulation of signaling in cellular response to high oxygen including some fluid balance changes responsible for the hematocrit decrease. Other interesting data are increasingly showing the interest of Antioxidant supplementation in Divers. (See references)

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